

### *Helicobacter pylori* (*H. pylori*)

*Helicobacter pylori* (*H. pylori*) is a gram-negative, spiral-shaped bacterium that impacts nearly 50% of the global population, particularly in developing nations. It is recognized as the primary contributor to chronic or atrophic gastritis, peptic ulcers, gastric lymphoma, and gastric carcinoma; however, these complications are less frequently observed in children and adolescents than in adults. The infection is typically contracted during early childhood and tends to persist if left untreated.<sup>1,2</sup>

There are four key factors that contribute to the development of clinical conditions such as gastritis and ulcers in the context of *H. pylori* infection.<sup>3,4</sup> Firstly, the urease activity of *H. pylori* is crucial for neutralizing the acidic conditions present in the stomach. Secondly, the motility of *H. pylori*, facilitated by its flagella, enables the bacterium to navigate towards the gastric epithelial cells of the host. This is succeeded by the interaction of bacterial adhesins with host cell receptors,<sup>5</sup> which is essential for effective colonization and the establishment of a persistent infection. Lastly, *H. pylori* releases various effector proteins and toxins, including cytotoxin-associated gene A (Cag A) and vacuolating cytotoxin A (VacA), which contribute to damage in host tissues.<sup>6</sup> In *H. pylori* gastritis, both acute and chronic inflammation is observed, characterized by the activation of eosinophils, neutrophils, mast cells, and dendritic cells.<sup>7</sup> Additionally, the gastric epithelial layer produces chemokines to trigger innate immune responses and activate neutrophils, which further exacerbate tissue damage, ultimately leading to the onset of gastritis and ulcers.

### References

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